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## Decompression Sickness, Extravehicular Activities, and Nitrogen Induced Osmosis: Brian Hills Revisited

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Decompression sickness has been recognized as an environmental and occupational illness for over 100 years, yet we still today are trying to find ways to minimize its effects or prevent the illness altogether. As the International Space Station is being built over the coming years, new challenges arise in attempting to manage the demands of physical labor in space without producing decompression sickness. The etiology of decompression sickness has evolved over the past century from Boyle and Bert to Haldane to many of those reading this article. Oftentimes, it is interesting to return to earlier work and research and see how it may apply to today's problems. The purpose of this paper is to take a historical perspective on one researcher of yesteryear, Dr. Brian Hills. It is not to advocate any changes in decompression sickness preventive measures.

First of all, who was Brian Hills? An Australian electrical engineer, he became a physiology researcher who studied the biological mechanisms of DCS during the 1960s and 1970s at Duke University and the University of Texas Medical Branch, Galveston. His work contributed to research programs at the Ministry of Defence, the US Navy, and the US Air Force to name a few. A prolific writer, he was the primary author of over 50 published scientific papers in addition to texts.

One text in particular is entitled Decompression Sickness: the Biophysical Basis of Prevention and Treatment (1977). In this text he developed elaborate mathematical model of DCS based on thermodynamics that was the result of many of his bench level studies. In it, he elaborates on many principals, some of which today are obvious. These include:

1. The primary event and the critical insult in producing symptoms of DCS do not coincide.
2. The primary event is the activation of one or more of a reservoir of nuclei normally present in tissue into growth and hence the inception of a stable gaseous phase.
3. Gas separates from solution in extravascular sites and in static blood.
4. Limb bends are determined by pressure distorting nerve endings and are more dependent upon the gas volume separated from solution.
5. No more than one anatomical type needs to be involved in marginal cases of limb bends.
6. Much larger volumes of gas are deposited in fatty tissues and can be released into the venous system as bubbles which remain asymptomatic unless they fail to be trapped by the lungs.

Less known is a hypothesis that he developed: gas induced osmosis influences the distribution of body water. In this hypothesis, Hills preformed an elaborate experiment that demonstrated that the differential in the concentrations of gases induces osmosis across gross tissue sections. Transient gradients of inert gases are caused by a change in pressure or in the concentration of the breathing mixture. A decompression will allow maintenance of extravascular inert gas concentrations resulting in an osmotic shift of water out of the blood and into the cell or extravascular space. This further distorts the tissue and exacerbates the gas phase effects seen symptomatically. In other words, for short periods following the start of a rapid change in pressure, there can be substantial differentials in gas concentrations between blood and tissue, not necessarily across the capillary wall, but across more remote diffusion barriers such as the cell membrane. The imminence of limb bends depends

upon the local pressure differential to bend a nerve ending which, in turn, is largely determined by the maximum volume of gas which can separate from solution in a unit volume of tissue. During a saturation state decompression, where there is a large reservoir of inert gas deep in tissue, this gas could exert a significant osmotic pressure tending to pull water out of the blood and so increase the tissue fluid pressure. Hills noted that regarding general fluid shifts, a reduction of extravascular fluid pressure is likely to decrease the threshold and even reverse a marginal bend. This he demonstrated clinically by administering low molecular weight dextran. Hills used the gas induced osmosis phenomenon to hypothesize the etiology of dry joints in extreme depth diving, the narcotic effect of the noble gases, and aseptic bone necrosis in diving.

It is not a surprise that DCS is a concern in both diving and EVAs. There are unique differences and concerns in the microgravity environment compared to the fluid environment. These include space adaptation syndrome, fluid shifts, and bone loss. There will be a considerable increase in the number of extravehicular activities expected to occur with the advent of International Space Station (ISS) construction. In turn, the likelihood of a decompression sickness case to occur increases proportionally. It brings up the important issues of how to treat a decompression sickness case versus a mission interruption and possible deorbit. What are ways to prevent or decrease the probability of decompression sickness from occurring? One question not asked is will it occur at all? Since the advent of space missions, no case of decompression sickness has been publicly acknowledged by US or former Soviet authorities. There have been occasional complaints of transient joint pains during EVAs while working, but these have been attributed to cramping or suit mechanical pressure and spontaneously resolved. There was an alleged pain only bends during a moon walk excursion. Is there a plausible explanation?

Thus far, what we are doing to prevent DCS seems to be working although we would all agree that it is probably only a matter of time before a case occurs. Decompression sickness preventive measures have been discussed at this meeting and include optimized pre-breathing schedules, exercise in general as well as potentially during the pre-breathing period, and overall fitness. Most of our data are based on earthbound experience and experiments. How does the microgravity environment change this, if anything? Could the microgravity environment be protective for decompression sickness? Current theories contend that adynamia or the lack of joint stress in the microenvironment versus gravity is a factor. Another is that the effect of weightlessness on the return blood flow from peripheral tissues improves denitrogenation and therefore decreases decompression sickness while in space. But does Hills' work also apply?

It is common knowledge that there is a fluid shift and diuresis over first 48 hours as the body equalizes fluid pressure levels. According to Hills', theory, this relative extravascular tissue water loss allows compensatory volume for osmosis of water into tissues and even larger gas phase bubbles during EVA decompression without producing symptoms due to nerve ending distortion. That is, the extravascular space, being decreased from the fluid shift, can now accommodate a bubble that would have to grow sufficiently large before exerting a nerve ending effect that would be symptomatic. It should be remembered that bubbles are present in the vasculature and tissues well before symptoms present, if at all.

If this is true, can we facilitate this protective effect in the event of an emergency before the 48 hour fluid shift has occurred? This brings us to what was coined the "Smart Body Hypothesis" at the International Congress of Aviation and Space Medicine in 1985. Simply, the hypothesis states that a body when exposed to a changed environment will adapt such that it will optimize its function within the new environment. Facilitating the expected end point will also modify transient effects seen during the adaptation. Hence, if we can get to the endpoint faster, then we would reach this protective state sooner. One could probably facilitate the fluid shift end point by administering 50 mg of hydrochlorothiazide upon orbit insertion. This is one of the first diuretics approved for the control of high blood pressure in aviators. This may allow for an emergency EVA with decreased DCS probability after the medication induced diuresis. In addition, it may decrease or eliminate space adaptation syndrome symptoms as the transient fluid shifts play a part in this condition.

As a corollary, what about the alleged DCS in the man on the moon? If one looks at the physiology occurring, there was always an adaptation period allowed for the moon's gravity. One would have to assume that there would be a fluid shift back into the extravascular space, although of a smaller degree than with earth's gravity. Hence, the excursion on the moon's surface would not have as much protective effect. Considering the workload during these excursions, there would be an increased risk of DCS compared to the microgravity environment.

The work done by Dr Brian Hills may be instrumental in understanding and predicting the extent of decompression sickness seen during extravehicular activities. If his hypothesis on gas induced osmosis is correct, then the microgravity environment of space may actually be protective once the fluid shift has occurred. In addition, there may be an adjunctive mechanism to decrease DCS probability in an emergent event or decrease or eliminate the symptoms of space adaptation syndrome by facilitating fluid shift and diuresis by administering hydrochlorothiazide upon orbit insertion. It should be noted that the authors of this paper recognize the importance to continue preventive measures for decompression sickness in the diving and space environments and future research efforts to decrease DCS incidence. This paper is not intended to discount or ignore standard procedures that are currently in use or will develop in the future.

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